Absence of *Twisted Gastrulation (Twsg1)* Limits the Population of Cranial Neural Crest Cells

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**Twsg1 affects craniofacial development**

- Craniofacial defects are a very common birth defect.
- *Twsg1* mutants have craniofacial abnormalities, including altered derivatives of the first branchial arch (BA1).
- Twsg1 is involved in craniofacial development by regulating bone morphogenetic protein (BMP) signaling in the medial region of BA1.

**Twsg1 impacts neural crest cells (NCCs)**

- NCC migrate from the posterior midbrain (mesencephalon) to the medial region of BA1 that gives rise to most of the mandible.
- Defects in BA1 may be due to defects in NCCs.

The goal of this project was to study NCC population in *Twsg1* mutants. The hypothesis was that the midbrain formation might be abnormal and/or NCC marker expression would be decreased.

**Results**

- Midbrain specification is normal in *Twsg1* mice at E8.5.
- Expression of NCC marker *Crabp1* is normal at E8-E8.5, but reduced in mesencephalic trigeminal neurons (MTN) at E9.5
- As shown by Sox10 expression, NCC generation is not reduced in *Twsg1* embryos at E8-E8.5

**Conclusions**

- The NCC population is not decreased at E8.0 or E8.5 but derivatives of NCCs are reduced at E9.5.
- The NCC population is likely being depleted at a point between E8.5 and E9.5, for example through increased apoptosis or reduced proliferation.

**Future Goals**

- Future research will directly assess proliferation, apoptosis, and BMP signaling in NCC populations in the absence of *Twsg1*.
- The NCC population will be studied between E8.5 and E9.5 so that the limiting of the population can be better understood.

**References**
